Carotid artery trauma: A review of contemporary trauma center experiences

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Purpose: **Many issues surrounding the management and outcome of carotid artery injuries remain controversial. The purpose of this study was to review a large contemporary experience with such injuries in the setting of designated trauma centers.**
Methods: **A statewide computerized trauma registry was used to identify all patients with injuries to the common or internal carotid arteries from October 1987 to June 1993. The records of 82 such patients were retrieved and analyzed.**
Results: **Overall mortality and stroke rates were 17% and 28%, respectively. Patients presenting with coma or shock had a particularly bad prognosis (50% and 41% mortality, respectively). Internal carotid injuries resulted in mortality and stroke rates of 21% and 41%, respectively, compared with 11% each for common carotid injuries. Patients with blunt injuries had a much higher stroke rate (56% vs 15%) but had lower mortality (7% vs 22%) than did patients with penetrating injuries. Airway compromise and associated injuries did not affect prognosis. Operative repair and percutaneous balloon occlusion had the best survival and functional outcomes.**
Conclusions: **Operative repair offers the best chances for recovery in all categories of patients regardless of injury mechanism. Ligation is useful only as a last-resort lifesaving effort. Shock and neurologic impairment are poor prognostic factors but should not negate repair. (J VASC SURG 1995;21:46-56.)**

Injuries to the carotid arteries constitute a particularly challenging subset of arterial trauma, because they have the potential to produce exsanguinating hemorrhage, suffocating airway compromise, and debilitating cerebral damage. Carotid artery trauma is, however, a relatively infrequent injury, occurring in less than 2 per 1000 patients with trauma who require hospital admission (North Carolina Trauma Registry). Most studies of civilian carotid injuries have addressed small series of patients[1-4] or have described a single center's experience spanning several decades,[5,6] during which significant changes have occurred in both operative and perioperative care. Because of limitations of previous studies several aspects of the management and outcome of these injuries have remained unresolved and have been the subject of controversy. Issues include the role of arteriography, the differences between blunt and penetrating injuries, the differences between internal and common carotid injuries, the results of nonoperative therapy, and the role of ligation. In addition the optimal treatment and expected outcome of patients presenting with a neurologic deficit or in coma remains unclear.

The purpose of this study was to address some of these issues through a review of the current management and outcome of injuries to the common and internal carotid arteries in the setting of designated trauma centers. The eight trauma centers in North Carolina participate in a computerized trauma registry, which was used to identify all patients with the diagnosis of carotid injury. The review covered the 6-year period from the inception of the trauma registry in 1987 to 1993.

PATIENTS AND METHODS

The North Carolina trauma registry (NCTR) is a cooperative undertaking of the eight designated trauma center hospitals in North Carolina, the four medical schools, and the North Carolina Office of Emergency Medical Services. The NCTR collects data on all patients with trauma (all age groups and

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all mechanisms of injury) who are hospitalized for at least 24 hours and all patients declared dead in the emergency department. For the purposes of the NCTR patients with trauma are defined as those patients with an International Classification of Diseases Supplementary Classification of Diagnosis (ICD-9-CM) code between 800 and 959.9. Data on patients with trauma were entered into a database with a microcomputer at each hospital and were validated on entry by the trauma registrar and the physician staff at each hospital. At intervals these data were sent to the NCTR Central Data Collection Agency at the University of North Carolina at Chapel Hill. Trauma Score and Glasgow Coma Scale (GCS) were computed on the patients' arrival to the emergency department. The Injury Severity Scores (ISS) were derived from the patients' ICD-9 Diagnosis Codes with a method previously described.[7]

One hundred twenty data points were collected on each patient. These included demographics, data regarding the mechanism of injury, scene and transport time, injury severity score, Glasgow coma scale, trauma score, emergency room interventions, blood transfusions, and up to eight diagnostic and five operative codes. Hospital length of stay, hospital charges, length of stay in the intensive care unit, duration of mechanical ventilation, status on discharge, and disposition information were also recorded.

Data collection began on October 1, 1987. At the conclusion of this study on June 30, 1993, 56,827 patients had been included in the database. The trauma registry was used to identify all patients sustaining injuries to the common or internal carotid arteries during that period (ICD-9 diagnostic codes of 900.00 to 900.03). Ninety-six patients were thus identified, and a small database consisting of this subset of patients was extracted from the NCTR. These data were used to derive general demographic information, disposition information as a function of status on presentation and treatment modality, and use and cost information. The effect of various treatment modalities on outcome was determined by dividing patients according to their mental status on admission and the treatment they received. The mental status information was derived from the GCS, where patients with GCS of 3 to 8 were defined as comatose. Three treatment groups were considered: nonoperative, operative ligation, and repair. The last group contained all patients who had the continuity of their injured carotid arteries restored with an operation regardless of the specific method of repair. Mortality and functional outcome were derived from the discharge disposition information with the assumption that patients discharged home had no significant functional impairment.

For more detailed information pertaining to the injuries such as their mechanisms, evaluation, management, and outcomes, the individual records of the patients identified by the database were retrieved where feasible. Eighty-two such records were available for review and form the basis for the subsequent detailed analysis.

Statistical analysis. Comparison of the differences in frequency of events among different groups was performed with chi square analysis.

RESULTS

NCTR Database Review

Demographics. The database review for the period of the study identified 96 patients with injuries to the common and internal carotid arteries. The mean age was 30 years. Sixty-six (69%) men and 30 (31%) women were studied. Twenty-two (23%) patients presented in coma (GCS < 8), 56 (58%) had a GCS greater than 8, and in 18 cases the GCS could not be determined from the database review. The mean injury severity score was 19.

Outcomes. The outcome results are summarized in Table I. No deaths occurred in patients with GCS greater than 8 (no coma) regardless of treatment modality. In contrast mortality was 50% in the 22 patients who presented in coma (GCS < 8). Operative repair was associated with the best survival and functional outcome. However, the GCS was unknown for 18 (19%) patients, which included deaths in both the ligation and repair treatment groups.

Resource utilization. For these 96 patients who were analyzed from the NCTR database health care resource utilization, including lengths of stay in the hospital and in the intensive care unit, and charges were also examined based on mental status at presentation and on outcome. The average hospital stay was 15.3 ± 17.3 days, with 6.8 ± 8.0 days spent in the intensive care unit. The mean duration of mechanical ventilation was 3.3 ± 4.8 days. Hospital charges for all patients with carotid injury averaged \$35,600 ± \$32,400. By comparison the mean lengths of stay in the hospital and in the intensive care unit for the entire trauma population in the registry were 12.6 ± 15 and 4 ± 6 days, respectively. Average hospital charges were \$20,700 ± \$30,000.

Patients who died because of their carotid injury tended to do so early in their course (mean hospital stay 3.5 ± 7.0 days for nonsurvivors vs 17.1 ± 17.7 days for survivors). Consequently hospital charges

Table I. Results of NCTR database review

			Home		*Nursing care*		*Deaths*	
Treatment group	*Arrival GCS*	*No.*	*No.*	*%*	*No.*	*%*	*No.*	*%*
No. operation	3-8	14	1	7	4	29	9	64
	9-15	23	15	65	8	35	0	0
Ligation	3-8	2	1	50	0	0	1	50
	9-15	9	7	78	2	22	0	0
Repair	3-8	6	2	33	3	50	1	17
	9-15	24	21	88	3	12	0	0
Total		78	47	60	20	26	11	14

GCS, Glasgow Coma Scale, *3-8,* Coma.
Eighteen patients with unknown Glasgow coma scale were not included.

were less in the nonsurvivors ($24,860 ± $28,900) compared with those of survivors ($36,900 ± $32,700). For survivors major differences in resource utilization were seen based on the outcomes of the injury (as inferred from the discharge disposition). For those discharged to some form of rehabilitation facility the average hospital stay was 36.3 ± 35 days compared with 11.2 ± 9.0 days in patients who were able to be discharged home. In a similar fashion hospital charges for patients discharged to a rehabilitation facility averaged $67,400 ± $75,000 compared with $26,600 ± $22,000 for patients who were discharged home.

Chart review

Demographics. The records of 82 patients could be retrieved and were reviewed. The mean age was 31 years (range 9 to 73 years). A total of 57 males and 25 females were studied. Forty patients were white, 38 were black, and four were of other racial backgrounds. Fifty-five (67%) injuries were penetrating, and 27 (33%) were blunt. Whereas blunt injuries were equally distributed between males and females (13 in males, 14 in females), penetrating injuries were four times more prevalent among males (44 vs 11) ($p = 0.003$ by chi square analysis). Most of the injuries in females (56%) were blunt, whereas in males more than three fourths (77%) were penetrating.

Thirty-five (43%) patients had a common carotid artery injury (seven blunt, 28 penetrating), and 47 (57%) had internal carotid artery involvement (20 blunt, 27 penetrating).

An additional six patients with isolated external carotid artery injuries were also reviewed. All of these injuries were due to penetrating trauma. Four of these patients had airway compromise on presentation and required acute intervention. Four of these patients were treated with flow interruption (three operative ligations and one percutaneous angiographic coil embolization). All six patients were discharged alive and well and were not included in further analysis or discussion in this article.

Fifty-two (64%) patients presented with no neurologic deficit, whereas 10 (12%) presented in an alert condition but with a major neurologic deficit. Twenty (24%) patients were comatose on admission.

Outcome. Table II summarizes outcome results as they relate to neurologic status on presentation and therapeutic intervention.

Fourteen of the 82 patients died for a mortality rate of 17%. Of these patients, 10 (71%) had presented in coma. Forty-five (55%) patients were discharged with no neurologic impairment, and twenty-three (28%) patients suffered strokes.

Twenty (24%) patients presented in coma; the vast majority (80%) had penetrating injuries. Half of these patients died, and another four (20%) had strokes. In contrast, only four (6%) of the 62 patients presenting with a normal sensorium died. Nineteen (31%) of these patients had strokes.

A striking difference in outcome was seen between injuries to the common carotid artery and those to the internal carotid artery. Indeed patients with injuries to the common carotid artery had stroke and mortality rates of 11% each (four of 35 patients each), whereas these rates for patients with injuries to the internal carotid artery were 41% and 21%, respectively (19 and 10 patients out of 47) ($p = 0.02$ by chi square analysis). All four deaths in patients with injuries to the common carotid artery resulted from exsanguination caused by penetrating trauma, whereas eight of the 10 deaths in the patients with injuries to the internal carotid artery were due to neurologic devastation.

Mechanism of injury. Because of their significant differences in presentation, management, and outcome, blunt and penetrating injuries were exam-

Table II. Outcome based on admission neurologic status and therapeutic intervention

			Alive & well		*Permanent deficit*		*Dead*	
Treatment group	*Neuro status*	*No.*	*No.*	*%*	*No.*	*%*	*No.*	*%*
No operation	Coma	6	1	17	1	17	4	66
	Neuro deficit	4	0	0	4	100	0	0
	Intact	17	7	41	7	41	3	18
Ligation	Coma	7	3	43	0	0	4	57
	Neuro deficit	1	0	0	0	0	1	100
	Intact	6	3	50	3	50	0	0
Repair	Coma	6	2	34	2	33	2	33
	Neuro deficit	5	2	40	3	60	0	0
	Intact	24	22	92	2	8	0	0
Balloon	Coma	1	0	0	1	100	0	0
	Neuro deficit	0	0	0	0	0	0	0
	Intact	5	5	100	0	0	0	0
Total patients		82	45	55	23	28	14	17

ined separately. Twenty-four of the 27 blunt injuries were the result of motor vehicle crashes, two were due to falls, and one was due to a crush injury. Forty-four gunshot wounds, seven shotgun blasts, and four stab wounds accounted for the 55 penetrating injuries.

Most of the penetrating injuries (66%) involved zone II (between the clavicles and the angle of the mandible). Penetrating injuries involved the common and internal carotid arteries with similar frequency (51% common carotid artery and 49% internal carotid artery). For the blunt injury group zones II and III were equally affected (44% and 41%, respectively); 74% of blunt injuries involved the internal carotid artery. All four patients with bilateral injuries were in this group, and three of them had strokes.

Of the 14 deaths in this series 12 were in patients with penetrating injuries. Mortality for the 55 penetrating injuries was 22%, whereas for the 27 blunt injuries it was only 7% ($p < 0.001$ by chi square analysis). However, the stroke rate for the blunt injuries was 56%, even though only 15% of these patients presented in coma and 18% presented with a deficit. Although it is a powerful predictor of mortality for patients with penetrating injuries ($p < 0.001$ by chi square analysis), the presence of coma on admission did not seem to correlate with mortality for patients with blunt injuries (zero of four) ($p = 0.75$ by chi square analysis), nor did it correlate with neurologic dysfunction for either group (two of 16 for penetrating injury and two of four for blunt injury). All five patients with blunt injuries who presented with a neurologic deficit short of coma were discharged with strokes. Additionally eight patients with blunt injuries who presented with no neurologic deficit had strokes. These patients had delays in the onset of their symptoms ranging from 3 hours to 3 days. Seven were treated without operation, and the one who underwent a surgical procedure was operated on 3 days after repair of an aortic transection and neck exploration for an esophageal injury, during which the common carotid artery disruption was missed. In these eight patients presenting without symptoms the initial neurologic manifestations were invariably severe, and the clinical course was always one of rapid deterioration leading to dense neurologic deficits.

Associated injuries and shock. Fifty-one (62%) patients had associated injuries (Table III). The most common concomitant injury in the penetrating group was esophageal perforation (18%), followed by vertebral artery injuries (11%) and thoracic and facial involvement (7% each). In the blunt injury group extremity fractures were the most common associated injuries (33%), followed by thoracic injuries (26%) and injuries to the head and face. Thoracic injuries included lung contusions, flail chest, and fractures of the clavicles, ribs, and sternum.

Cranial nerve injuries were present in 21 (26%) patients. Patients with blunt injuries were slightly more likely to have cranial nerve damage than were those with penetrating trauma (30% vs 24%). Of the 16 concomitant internal jugular vein injuries, only one was in a patient with blunt trauma. The other 15 (27%) were in the penetrating group.

Of nine patients with concomitant vertebral artery injuries, four (44.5%) had strokes, and one (11%) died.

The presence of associated injuries did not seem to affect outcome ($p = 0.81$ by chi square analysis), but shock (systolic blood pressure < 90 mm Hg) did.

Table III. Associated injuries by decreasing order of frequency

Injury	*Blunt*	*Penetrating*	*Total*
Esophageal perforation	1	10	11
Thoracic injuries	7	4	11
Extremity fractures	9	0	9
Vertebral artery	3	6	9
Facial fractures	5	4	9
Skull fractures	6	2	8
Brain	4	3	7
Spinal fractures	3	2	5
Abdominal injuries	3	1	4
Soft tissue	1	3	4
Spinal cord	0	3	3
Other arteries	2	1	3

Of 22 patients presenting in shock, 20 sustained penetrating injuries. Patients presenting in shock had a mortality of 41% as opposed to 8% in the absence of shock ($p = 0.002$ by chi square analysis). The presence of shock did not affect neurologic outcome.

Angiography. Angiographic examination was performed in all 27 patients with blunt injuries. Patients with penetrating injuries were almost evenly divided in that regard (56% with and 44% without angiograms), with the presence or absence of shock on presentation being the major determinant. In the absence of shock 77% of patients (27 out of 35) with penetrating injuries underwent angiographic examination. This ratio was reversed in the presence of shock where only 20% of these patients (four out of 20) were studied with angiography ($p < 0.001$). Eighty-five percent of patients with zone I and III injuries had angiograms, whereas only 60% of patients with zone II injuries underwent angiographic examination. Angiographic findings for blunt and penetrating injuries are summarized in Table IV.

Airway compromise. Twenty-three (28%) patients had airway compromise requiring emergency measures. The vast majority (79%) had zone II penetrating injuries. The airway was compromised in 47% of patients in this group. Of these, five (29%) required tracheostomies, whereas the remaining 12 were treated with emergent endotracheal intubation. Of 28 patients with cervical hematomas, 17 (61%) had airway compromise, whereas only one (5%) of 20 patients without hematoma had airway compromise ($p < 0.001$). Despite the need for emergency airway treatment in more than one third of patients with zone II injuries, no adverse effect on survival or neurologic outcome was observed.

Management. To determine the influence of treatment on outcome, patients were divided into four management categories: nonoperative treatment (27 patients), ligation (14 patients), percutaneous balloon or coil occlusion (six patients), and repair (35 patients). The repair group consisted of those patients who had vascular continuity of their carotid arteries restored by primary repair (resection and anastomosis or lateral suture) (16 patients), patch angioplasty (five patients), or bypass (14 patients). Of 55 penetrating carotid injuries 28 (51%) were repaired, 17 (31%) were occluded (12 by operative ligation and five by percutaneous techniques), and only 10 (18%) received no operation.

The outcomes of the four different treatment modalities are depicted in Table V. The differences in treatment were most striking when patients presenting with intact neurologic status were examined (Table II). Twenty-two (92%) of 24 such patients were discharged alive and well after repair, as opposed to seven (41%) of 17 such patients treated without operation ($p = 0.005$ by chi square analysis). The six patients with intact neurologic status who were treated by ligation fared somewhat better than did patients treated without operation, with three (50%) being discharged intact. For those patients presenting with intact neurologic status no deaths occurred in either operative group (ligation or repair) compared with an 18% mortality rate for the nonoperative group. Moreover the stroke rate for the repair group was 8% as opposed to 41% and 50% for the nonoperative and ligation groups, respectively. The six patients treated with detachable balloon or coil occlusion had distal internal carotid artery injuries in zone III. All but one had carotid cavernous fistulas. All five patients with intact neurologic status who were treated with these percutaneous interventions made full recoveries.

Even though the numbers were small, similar trends were found for patients presenting in coma or with neurologic deficits. The mortality rate for the six

Table IV. Angiographic findings by mechanism of injury (15 patients had more than one finding)

Finding	*Blunt*	*Penetrating*	*Total*
No angiogram	0	24	24
Pseudoaneurysm	5	12	17
Dissection	14	1	15
Occlusion	1	6	7
Carotid cavernous fistula	1	4	5
Avulsion/transection	2	2	4
Extravasation	1	3	4
Intimal injury	2	1	3
Negative	1	1	2
Intraluminal clot	0	1	1
Total	27	55	82

comatose patients treated without operation was twice that of the six who underwent a surgical procedure (66% vs 33%). Only one of these patients was discharged alive and well. All four patients who presented alert but with a neurologic deficit, and had not undergone a surgical procedure had strokes. However, two of five patients with similar presentations who underwent repair were discharged alive and well. Because these numbers are too small, the clear trend favoring repair could not be validated statistically in these groups.

Stated in somewhat simplistic terms the overall combined mortality and stroke rates were 70% and 57% for the nonoperative and ligation groups, respectively. These rates were 26% and 17% for the repair and balloon embolization groups, respectively (Fig. 1).

DISCUSSION

Because of the complexity of carotid injuries and the multitude of factors and considerations affecting therapy and outcome, a relatively large number of case reports and review articles have dealt with the subject over the past 30 years.[1-6,8,9] Controversies regarding the evaluation and management of these injuries have not been settled. In this study we have attempted to overcome some limitations of previous studies and perhaps to enlarge upon the experience of others by using a State Trauma Registry that has provided information on a sizable number of patients collected during a relatively short period of time. This has resulted in a synchronous and contemporary experience. We also took advantage of the database to touch upon an issue that is becoming increasingly relevant, which is healthcare resource utilization and cost.

This computerized registry can provide a wealth of information on large numbers of patients and allows rapid processing and analysis of huge amounts of data. It is most useful in drawing broad conclusions, discerning trends, and identifying patient subsets of interest that warrant closer examination with conventional chart reviews. The information derived from the database should be viewed with its limitations in mind. Despite ongoing quality control checks by the trauma registrars and physician staff at each institution, some errors are inevitable. These can range from clerical errors to differences in interpretation and coding of data to be entered. Additionally we have learned that certain types of information, those pertaining to angiograms being an example, are unreliable. Another major limitation has been missing information that may be extremely relevant to the issue at hand. A clear example is the lack of GCS information on 19% of the patients in our NCTR review. Thus the database is more of a descriptive tool than an analytic one.

The database review underscored the dire consequences of carotid injuries in general. The mean hospital length of stay and charges were 3 days longer and three times higher, respectively, for carotid injuries compared with those of the overall trauma population. The review also demonstrated the ominous prognosis of coma on presentation and the benefits of arterial repair in the management of these injuries. However, more information was needed; hence the individual record review was done. In spite of its advantages over the database review the individual record review remains a retrospective one with all the limitations inherent to such endeavors. The fact that all the patients were cared for in designated trauma centers within one state and for a relatively short period of time accounts over the similarity and consistency in approach to diagnosis and management that were seen. However, this is still far from the uniformity of diagnostic and treatment protocols afforded by prospective clinical trials.

Table V. Outcome by treatment modality

		Alive & well		*Stroke*		*Dead*	
Treatment	*No.*	*No.*	*%*	*No.*	*%*	*No.*	*%*
No operation	27	8	30	12	44	7	26
Ligation	14	6	43	3	21	5	36
Repair	35	26	74	7	20	2	6
Balloon	6	5	83	1	17	0	0
Total patients	82	45	55	23	28	14	17

The record review further confirmed the dismal prognosis of comatose patients. Additionally it revealed significant differences between injuries to the common and internal carotid arteries. Injuries to the internal carotid artery were associated with a markedly worse neurologic prognosis and mortality than were injuries to the common carotid artery. Whereas most deaths caused by internal carotid artery injury were neurologic, all four deaths caused by common carotid artery injury were due to uncontrollable hemorrhage.

Shock had a deleterious effect on survival but did not seem to affect neurologic outcome. The presence of associated injuries (62%) and airway compromise (28%), though common, did not affect outcome. Esophageal perforation was relatively frequent in patients with penetrating injuries and should be specifically looked for in these patients. The dire consequences of a missed esophageal injury adjacent to an arterial repair were vividly demonstrated in one of the patients in this series. Our experience with nine concomitant vertebral injuries showed no deleterious effect on survival (one death) but only a trend toward increased stroke risk. This finding is different from the Parkland experience reported by Landreneau et al.,[10] where eight patients with combined carotid-vertebral injuries had a 50% mortality rate.

Internal jugular vein injuries, which were present in 27% of patients with penetrating injuries, did not affect outcome. Only one such injury was seen in the group with blunt trauma.

Blunt injuries were encountered much more frequently than previously reported (one third of the patients)[3,11-13] and were mostly due to motor vehicle accidents. This finding may be the result of increased recognition through more aggressive use of angiographic examination, rather than a truly higher incidence of this elusive lesion. Also North Carolina is still a predominantly rural state and may not yet reflect the high incidence of penetrating violence reported from urban areas. Mortality rates for patients with blunt injuries were much lower (7%) than were those for patients with penetrating injuries in our series and those for patients with blunt injuries previously reported by others. Indeed Krajewski and Hertzer[14] and Perry et al.[3] reported mortality rates of 30% and 23%, respectively. Unfortunately although two thirds of patients with blunt injury to the carotid artery presented neurologically intact, more than half were discharged with significant neurologic deficits. This occurrence was most certainly due to the delay in diagnosing these injuries until after the development of symptoms, which was hours to days after injury. Patients with blunt injuries were more likely to be treated without operation than were those with penetrating injuries (63% vs 18%) and had a higher incidence of associated injuries, which may have confounded their evaluation and management.

Angiography is extremely helpful in the current management of carotid injuries. This fact is well recognized as indicated by the liberal use of angiographic examination in all patients with blunt injury and in an overwhelming majority of patients with penetrating injury who were hemodynamically stable. Patients with hemodynamic compromise as a rule were taken to the operating room expediently without angiographic examination. Of the four exceptions three were discharged alive and well, and one had a stroke while in the angiography suite. Angiograms were obtained much more frequently in zone I and III injuries, where they are most useful. In stable patients with zone II injuries, angiograms can be helpful in minimizing unnecessary surgical explorations and, more importantly, in reducing the chances of missing existing injuries.

Duplex scans were obtained in only three of the 82 patients. This low rate may be due to the limited availability of duplex scans at odd hours and on short notice and the limited need for them in most zone II injuries, where they would most likely be used. One exception would be occult blunt injuries. Indeed duplex scanning may be greatly underused in that setting and may have a role to play in screening for these elusive injuries in an effort to make a timely

diagnosis before the onset of neurologic symptoms. Because a significant number of patients with blunt injury in this series presented intact and then had fixed deficits once symptoms developed, it would not be unreasonable to screen all patients with significant blunt trauma to the head and neck, much like the way screening for cervical spine injuries is done. Prospective evaluation of such an approach in trauma centers with large volumes of blunt injuries is needed.

Ambroise Paré[15] treated a carotid injury in a duelist by ligation in 1552 with an outcome of aphasia and left hemiplegia. In 1798 Abernathy ligated a carotid artery for hemorrhage in a patient gored by a bull, but the patient died 30 hours later. The first successful carotid ligation is credited to David Fleming,[16] a captain on the H.M.S. Tonnant, who in 1803 "cut down upon and tie(d) the carotid artery below the wound" in a sailor attempting suicide. Ligation remained the principal form of therapy for the remainder of the nineteenth century and the first half of this century. Cerebral complication rates of 30% to 33% and mortality rates of 45% to 50% continued through World War I and World War II.[17,18] With the advent of modern vascular techniques and rapid triage in the Korean war, operative repair became acceptable and appeared to lower the mortality rate to 10%.[17] The Vietnam war brought improved outcomes mainly because of further refinements in triage, rapid transportation, and resuscitation. However, another swing of the pendulum occurred in 1970, when Cohen et al.,[19] reporting on their experience in Vietnam, suggested that restoration of vascular continuity was directly responsible for the deaths of some of their patients who had presented with neurologic deficits. Based on Wylie's work with patients undergoing revascularization for acute thrombotic strokes,[20] Cohen et al. postulated (without autopsy evidence) that revascularization of neurologic deficits was converting anemic infarcts into hemorrhagic ones. They concluded that ligation may be the treatment of choice for "many" patients with neurologic impairment. Bradley's 1973 report of two patients who presented with preoperative neurologic deficits and who were found to have hemorrhagic cerebral infarctions on autopsy after repair of their carotid injuries further strengthened the argument for ligation of the carotid arteries in patients with neurologic impairment.[21] The controversy has been raging since then.

During the past 2 decades numerous studies[4-6,22] have revisited the issue and have suggested a definite benefit for revascularization, when feasible, in all patients with carotid artery injuries. The recognition

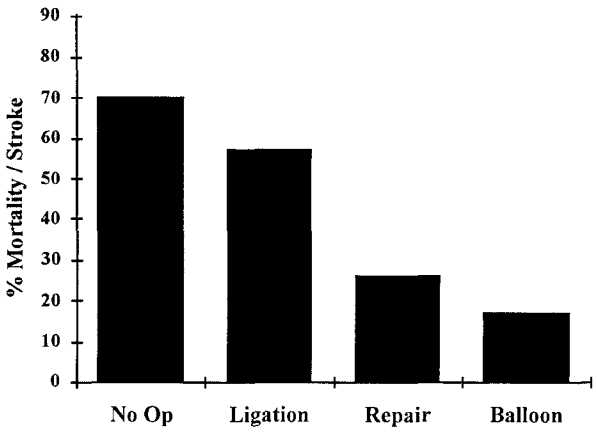

Fig. 1. Combined mortality and stroke rates as function of treatment modality.

by a number of authors such as Weaver et al.[9] and Ledgerwood et al.[23] that neurologic deterioration after repair is the result of cerebral edema and not hemorrhagic infarction has further strengthened the argument for repair. It has become clear that extrapolation from observations made in elderly patients who have undergone revascularization after atherosclerotic strokes[20] to those made in the young trauma population is hardly valid.[4,23]

Our data strongly support an aggressive approach towards operative intervention and repair for all patients with carotid injuries when feasible (Fig. 1). For patients presenting with intact neurologic status, the benefit is clear (Table II). For our patients who presented in coma, mortality was twice as high in the nonoperative group compared with that in the repair group. We agree with Brown et al.,[5] Weaver et al.,[9] Richardson et al.,[24] and Fabian et al.[25] that repair should be attempted in all patients, even those in coma. First the severity of the neurologic deficit is not always possible to ascertain. The depressed mental status may be due to shock, intoxication, sedation, paralysis for airway control, or often a combination of these. Second strong evidence exists in the literature that the ischemic insult, rather than reperfusion hemorrhage, is the major cause of untoward outcome in these patients.[9,23] The concept of limiting ischemic damage by reperfusing the "ischemic penumbra" around an area of irreversible infarction[26] may account for the clinical observations of improvement seen after restoration of flow in certain patients with preoperative neurologic deficits. Finally in an excellent review of 722 cases, Unger et al.[22] found no evidence to support coma as a contraindication to the repair of carotid injuries. Although clearly a poor

prognostic sign, the presence of coma should not contraindicate repair. We agree with Brown et al.[5] that more emphasis should be placed on aggressive management of postischemic cerebral edema and the provision of optimal cerebral blood flow, which have the potential benefit of limiting infarct size and hence reducing the magnitude of the neurologic dysfunction, rather than denying these patients revascularization.

Of the 24 patients who presented with intact neurologic status and underwent repair, only two had strokes. One had a stroke before the operation in the angiography suite. The other had a missed common carotid artery injury for 3 days despite neck exploration at the referring hospital for repair of an esophageal tear. Shunts were used infrequently (five patients only). All were in patients with common carotid injuries, and all were intact on presentation. One patient had a stroke, which occured in the radiology department while the patient was undergoing angiographic examination. Because all the other patients did well without them, the need for shunts in this setting remains uncertain.

Ligation was a lifesaving compromise. None of our patients who presented with intact neurologic status and were treated with ligation died. However, three of six had strokes. Overall 43% of the patients who underwent ligation were discharged alive and well, which was a better result than in the nonoperative group but was far inferior to the repair group. Ligation was mostly used as a last resort in situations where repairs were impossible, usually because of uncontrollable hemorrhage, hemodynamic instability, and in one instance breakdown of a repair from infection resulting from a missed esophageal tear. This is reflected in the high mortality rate in this group.

In a select group of patients, namely those with high zone III internal carotid artery injuries, detachable balloon or coil occlusion had excellent results. This is the modality of choice in patients with carotid-cavernous fistulae and other carefully selected patients with high inaccessible injuries.[27]

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DISCUSSION

Dr. Daniel J. Reddy (Detroit, Mich.). The authors' use of the North Carolina Trauma Registry of patients with carotid injury offers certain advantages over a single-center report. For example, all patients discharged from the eight participating institutions with this uncommon and often complex diagnosis were combined, making a large series analyzed in a standard fashion.

The relatively brief time period dampens the changes in trauma resuscitation, transport, or surgical management that otherwise would have been inevitable during the decades-long experience needed for a comparably sized single-institution report. Also the outcome of both nonoperative and a variety of operative management schemes pursued by the same trauma teams are compared.

Though carefully analyzed, up to 18% of the data points are missing from this retrospective study, and this patient cohort remains noticeably heterogenous for its size. By inference it cannot include all patients with carotid injury, because the authors have demonstrated that many blunt carotid injuries are initially silent and are only discovered when actively searched for by angiographic examination, or when a delayed neurologic deficit develops after hospital admission. A prospective evaluation of all patients for carotid injury would likely have revealed a higher incidence of carotid blunt injuries that would have remained silent. These undiscovered carotid injuries may have altered the conclusions regarding blunt injury.

Foremost among the many important conclusions of this study is the demonstration that though coma on admission is a powerful predictor of mortality after penetrating injury, it correlates poorly with mortality for blunt injury. Moreover it does not correlate with stroke in either group. Some confusion in the literature results from the blurring of this distinction between coma and stroke by simply categorizing patients as either normal or neurologically impaired. Today the authors have clearly considered the prognostic difference between coma and stroke and have made the case for aggressive approach in the management of carotid lesions, both in the neurologically intact patients and those in coma. In addition they have clarified the selective use of interventional radiographic techniques in zone III carotid cavernous fistula injuries and the occasional need for carotid ligation as a lifesaving compromise.

I would like to ask Dr. Ramadan the following questions. Do you agree with other authors advocating anticoagulation as the primary therapy for blunt injuries resulting in carotid dissection? Do you have autopsy or medical examiner findings regarding the pattern of brain edema, infarction, hemorrhage, or embolization in the 13 deaths? Does duplex scanning rule out blunt injuries in zone II? Can it be recommended as a screening test to limit angiography? What do you recommend for the minimal carotid intimal defect? Is nonoperative follow-up of intimal defects unduly hazardous in the carotid artery? Some have hypothesized that blunt carotid injury results from either a stretching of the carotid artery over the transverse processes of C2 and C3 or by direct carotid compression between the mandible and these processes. What can you tell us about the mechanism of blunt injury? Finally, what is your recommended treatment for a zone II injury in a stable and alert patient who has, however, a focal neurologic deficit? What is the role of angiography or operation in such a patient?

And again, I very much enjoyed this timely, well-researched, and well-presented article and intend to be guided by its conclusions.

Dr. Fuad Ramadan. As far as the role of anticoagulation no homogeneous approach was made to patients with blunt injuries that were treated conservatively. Patients were split about 50-50 as far as the use of aspirin and the use of real anticoagulation with heparin and then coumadin, so we could not make any conclusions in that regard.

We do not have any autopsy data that address the question of hemorrhagic infarction or brain edema, and our discussion in the article was mainly drawn from series in the literature.

As far as diagnosing blunt injuries with duplex scanning, the article raises that issue. One of our conclusions or recommendations is that patients who have severe head and neck injuries should perhaps be screened routinely, the same way patients with those injuries are screened for C-spine fractures. I think it may be a cost-effective thing to do and is certainly worth studying in a prospective fashion.

As far as the management of minimal defects, we had quite a few examples that time did not permit us to discuss,

where minimal defects were treated conservatively under close observation and did resolve.

As far as the management of zone II injuries in stable patients with a deficit, we looked at the relation of angiography to the outcome, and in all instances except for one no harm was done by getting an angiogram before operation. Only one patient who presented with intact neurologic status and who had a zone II injury had a stroke in the arteriogram suite, but by and large no harm was done in those patients who underwent angiography for zone II injury, and I think it may be helpful to obtain that if the conditions permit.

Dr. Padberg. The authors have again raised many of the controversial questions in management of multietiologic carotid trauma. Their mix of patients includes 25% blunt trauma, a group in which it is difficult to separate the contribution of head and cervical injury from that caused by arterial injury. Forty percent of the injuries were repaired. Although changes in trauma care delivery may have occurred recently, management recommendations have changed very little and may hinge on the neurologic assessment. For the authors to reiterate that coma was a strong predictor of survival confirms many previous reports on this subject. In our experience the response to simple fluid resuscitation in profoundly hypertensive patients may dramatically improve the initial neurologic score before operation.

Our report to the Eastern Vascular Society last year incorporated a 12-year experience from our institution and a 1316-patient literature review of penetrating carotid injury (J VASC SURG 1993;18:318). Based on this experience repair was significantly better for those with no deficit. In addition it was significantly better for those with focal or lateralizing deficits. Patients with a reduced state of consciousness defined as a Glasgow Coma Scale from 9 to 14 were also best served by repair, although the numbers were small. Thus we have recommended that patients with a Glasgow Coma Scale greater than 8 should be considered for surgical repair when feasible. A trend toward improved outcome with repair was also observed with comatose patients, although statistical significance was not achieved.

I have several questions. The authors have stratified patients according to whether they were comatose on arrival. Because aggressive resuscitation may improve cerebral symptoms, how many of these patients changed their global neurologic scoring after the admitting assessment?

Second, what was the impact of the Glasgow Coma Score in their surgical treatment, for example, for those patients who had scores less than 8 or for those who had scores in the 9 to 14 range?

Dr. Ramadan. Thank you, Dr. Padberg, for sharing your abstract from last year with us. Your point is very well taken as far as the overall hemodynamic status of the patients affecting their Glasgow Coma Scale. That is why we did not put a lot of faith in the actual Glasgow Coma Scale that was seen on admission. Patients can be comatose because of hypotension, brain injury, alcohol, and the carotid injury. We did go over the neurologic examination of each and every patient in detail.

As far as the Glasgow Coma Scale or at least the neurologic examination changing, most of the patients we encountered had worsening of their neurologic status rather than improvement from the time of admission to the time of intervention or to the time of discharge. That was one of our concerns, and that is why we looked beyond the simple number of the Glasgow Coma Scale.

Dr. Thomas S. Riles (New York, N.Y.). Very simply, it seems as though you have concluded that operation improves the outcome, but the patients who were not operated in this series must have been the worst at the time of presentation. Maybe you could tell us why it was that you did not operate on those particular patients.

Dr. Ramadan. Again, this is a retrospective study, but we did look to see whether a relation existed between the mode of treatment and the presentation mental status, and no correlation between the two was found. I did not show those data because of time constraints, but again, no correlation was seen between the mode of therapy chosen and the neurologic status on admission.

Dr. Martin G. Veller (Johannesberg, South Africa). We recently reviewed our experience with internal carotid artery injuries, and these were largely penetrating injuries as a result of stab wounds, and we found that if the patients survived the initial therapeutic intervention, we came across a large number of complications as a result of pseudobulbar palsy-associated nerve injuries in this area, and this in fact accounted for about 50% of our deaths. What is your experience in this field, and did you have a similar kind of problem?

Dr. Ramadan. Actually, for surviving patients the mean hospitalization was 11 days, and we encountered about a 21% rate of associated cranial nerve deficits or local nerve deficits, but this finding did not seem to have an impact on survival. None of the associated injuries seemed to affect survival. Shock was the only variable that really had a significant impact on the outcome.